



NOTES

MALABSORPTION CONDITIONS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Impaired ability of gastrointestinal tract to absorb nutrients
- Malabsorption may be
 - Global → impaired function of intestinal cells
 - Partial → external agent interferes with absorption
- Manifestation of underlying illness; may be congenital/acquired/infectious

CAUSES

- Defects in absorption process of intestinal cells (e.g. change to bowel surface area)
- Impaired nutrient digestion (e.g. altered digestive enzymes)

SIGNS & SYMPTOMS

- Abdominal distention, pain
- Chronic diarrhea, malabsorption, dehydration
- Weight loss
- Clinical manifestations of nutritional deficiencies (e.g. paresthesias from cobalamin deficiency)

DIAGNOSIS

DIAGNOSTIC IMAGING

- Abdominal ultrasound, colonoscopy, intestinal biopsy, serological markers

LAB RESULTS

- D-xylose test
 - Test for carbohydrate malabsorption
- Fecal fat testing
- Complete blood count (CBC)
 - Look for infection, anemia

OTHER DIAGNOSTICS

- Individual history
 - Pancreatitis
 - Recent surgeries
 - Symptoms of vitamin deficiency
 - Family history

TREATMENT

- See individual disorders

VISIT...

LANZAROTE
Caliente.COM

CELIAC DISEASE

osms.it/celiac-disease

PATHOLOGY & CAUSES

- Autoimmune disorder of small intestine
- AKA gluten-sensitive enteropathy/nontropical sprue

CAUSES

- Triggered by foods containing gliadin, a peptide found in foods containing gluten (e.g. grains: wheat, barley, rye, oats)
 - Gluten consumption → degradation into peptides in small intestine → secretory IgA binds to gliadin in duodenum → IgA-gliadin complex binds to transferrin receptor → IgA-gliadin complex travels across enterocyte into lamina propria → tissue transglutaminase deaminates gliadin → macrophages uptake, present deaminated gliadin in MHC-2 molecules HLA-DQ 2, 8 → CD4+ activation → inflammatory cytokines released (interferon gamma, tumor necrosis factor) → damage/destruction of intestinal villi → B cell activation → anti-gliadin, anti-tissue transglutaminase, antiendomysial antibodies released → CD8+ cell activation → tissue destruction
- On microscopy
 - Villous atrophy, mucosal inflammation, intestinal crypt hyperplasia
- Presence of anti-gliadin, anti-endomysium IgA = pathognomonic

RISK FACTORS

- Northern European ancestry, genetic component

SIGNS & SYMPTOMS

- Abdominal distention, chronic diarrhea (steatorrhea)
- Failure to thrive (children)
- Dermatitis herpetiformis
 - Circulating IgA antibodies attack dermal papillae → generalized rash

DIAGNOSIS

LAB RESULTS

- Anti-gliadin IgA/IgG
- Anti-endomysium IgA
- Anti-tissue transglutaminase IgA
 - Tissue transglutaminase: endomysial enzyme released in response to cellular stress
 - More sensitive, specific

Duodenal biopsy

- Showing lymphocytic infiltration, villous atrophy, crypt hyperplasia

TREATMENT

OTHER INTERVENTIONS

- Correct nutritional deficiencies related to malabsorption

Preventative

- Gluten-free diet



MNEMONIC: Grains are BROWN

Grains to avoid with Celiac disease

Barley

Rye

Oats

Wheat

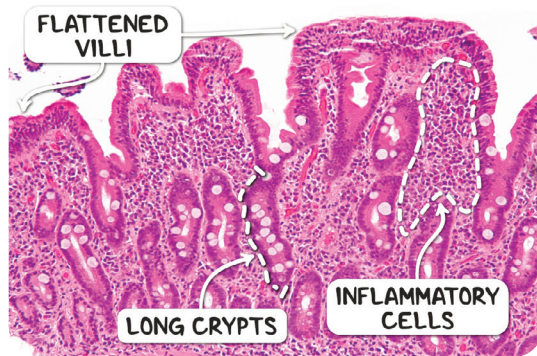


Figure 38.1 Histological appearance of a duodenal biopsy in an individual with celiac disease. There is villous blunting, an expansion of the lamina propria by chronic inflammatory cells and an increase in crypt length. A higher magnification would reveal an increase in lymphocyte count in the surface epithelium.



Figure 38.2 Clinical appearance of dermatitis herpetiformis. Individual with celiac disease are at increased risk of this condition.

LACTOSE INTOLERANCE

osms.it/lactose-intolerance

PATHOLOGY & CAUSES

- Decreased ability to digest lactose
- Lactose consumption → lactase deficiency/inactivity → ↑ undigested lactose → fermentation by colonic flora → gas, osmotically active substances produced → bloating, diarrhea

CAUSES

- Most often acquired due to physiologic weaning off of milk

RISK FACTORS

- Non-European ancestry (most common)
- Increases with age
- May be congenital
 - Rare, autosomal recessive disorder
- May be developmental
 - Most common among premature infants
- Underlying intestinal disease

SIGNS & SYMPTOMS

- Occur after consuming lactose (e.g. milk, cheese)
- Abdominal pain, cramping in lower quadrants
- Abdominal distention, flatulence, vomiting, diarrhea (more common in children)

DIAGNOSIS

- Based on above symptoms

LAB RESULTS

- Unabsorbed carbohydrates → high stool osmotic gap
- Bacterial lactose fermentation → acidic stool pH

TREATMENT

OTHER INTERVENTIONS

- Optimize calcium, vitamin D intake

Preventative

- Lactose-free diet
 - Compensate with lactase

SMALL BOWEL BACTERIAL OVERGROWTH SYNDROME

osms.it/sbbos

PATHOLOGY & CAUSES

- Excessive colonic bacteria colonizing small intestine
- Often occurs secondary to conditions limiting intestinal motility, gastric acid and bile secretion and IgA deficiencies

CAUSES

- Alteration of factors regulating intestinal flora → aerobic bacteria proliferation → changes in aerobic microclimate of small intestine → migration of colonic anaerobic bacteria → damage to intestinal surface → maldigestion, malabsorption → symptoms
 - ↑ bacteria → ↑ carbohydrate metabolism → ↑ gas production → bloating
 - ↑ bacteria → bile acid inactivation → ↑ fat in colon → osmotic effect → diarrhea
 - ↑ bacteria → intrinsic factor degradation → impaired B₁₂ absorption → B₁₂ deficiency

RISK FACTORS

- Increases with age

SIGNS & SYMPTOMS

- Abdominal pain/distention, chronic diarrhea, flatulence
- Tympanitic abdomen upon percussion

- Altered mental status after high carbohydrate meal
- Failure to thrive (children)

DIAGNOSIS

LAB RESULTS

- Signs/symptoms of vitamin, electrolyte abnormalities
 - Weakness, ataxia, paresthesias → B₁₂ deficiency
 - Perioral numbness, feet paresthesias, muscle cramping → calcium deficiency
- Anemia
 - Macrocytic → B₁₂ deficiency
 - Microcytic → chronic bleeding
- Fecal fat testing
- Lactulose/glucose breath testing
- Jejunal aspirate, culture
 - > 10³ colony forming units

OTHER DIAGNOSTICS

- Individual history
 - Chronic pancreatitis, intestinal surgery, GI neuropathy

TREATMENT

MEDICATIONS

- Antibiotics

TROPICAL SPRUE

osms.it/tropical-sprue

PATHOLOGY & CAUSES

- Gastrointestinal disease of uncertain etiology resulting in **nutrient malabsorption**

CAUSES

- Acute intestinal infection (viral/bacterial/protozoan) → damaged intestinal lining → inflammation → enteroglucagon secretion → decreased intestinal motility → increased intestinal transit time → overgrowth of *Klebsiella*, *E. coli*, *Enterobacter* → production of toxic fermentation byproducts → further inflammation → villous atrophy → malabsorption → depletion of folate, B₁₂ → intestinal villi can't function normally → further intestinal injury, megaloblastic anemia

RISK FACTORS

- Most common in individuals living in **tropical regions**

SIGNS & SYMPTOMS

- Diarrhea, weight loss, dehydration, abdominal pain, fatigue, megaloblastic anemia

DIAGNOSIS

DIAGNOSTIC IMAGING

Endoscopy

Barium swallow

- Shows intestinal wall thickening

LAB RESULTS

- Fecal fat test
- D-xylose test
- Jejunal biopsy
 - Shows villous atrophy, inflammation

TREATMENT

MEDICATIONS

- **Antibiotics** → reduce bacterial overgrowth
- Replace folate, B₁₂

WHIPPLE'S DISEASE

osms.it/whipples-disease

PATHOLOGY & CAUSES

- Rare, malabsorptive infectious disease caused by *Tropheryma whipplei*
- Pathognomonic finding → lamina propria displays numerous macrophages with periodic acid-Schiff (PAS) positive intracellular material

CAUSES

- *Tropheryma whipplei*
 - Gram-positive, non-acid fast, PAS positive bacillus; ubiquitous in environment
 - Fecal-oral transmission
- Readily spreads throughout body, causing multisystem effects
 - Evades immune response → allows for accumulation of bacilli in tissues
- Current hypothesis suggests host immunodeficiency as predisposing factor

RISK FACTORS

- Middle-aged biological males of European ancestry; exposure to fecal matter (sewage workers, farmers)



MNEMONIC: WHIPPLES

Features of Whipple's disease

Weight loss

Hyperpigmentation of skin

Infection with *tropheryma whippelii*

PAS positive granules in macrophage

Polyarthrititis

Lymphadenopathy

Enteric involvement

Steatorrhea

SIGNS & SYMPTOMS

- Four cardinal signs
 - Diarrhea, weight loss, abdominal pain, arthralgias
- Endocarditis, pericarditis, myocarditis
- Skin hyperpigmentation
- Pleural disease

DIAGNOSIS

LAB RESULTS

- Biopsy
 - Shows copious PAS positive macrophages invading lamina propria in intestine
- ≥ two positive PCR/PAS tests
- Immunohistochemistry for *T. whipplei*
- Laboratory findings suggesting chronic inflammation, nutritional deficits

TREATMENT

MEDICATIONS

- Start with IV antibiotics → ceftriaxone/penicillin G
- Trimethoprim-sulfamethoxazole (1 year)

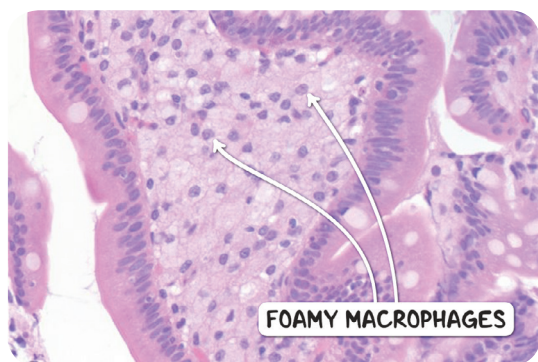


Figure 38.3 Histological appearance of the duodenum in a case of Whipple's disease. The lamina propria is occupied by numerous foamy macrophages. Electron microscopy would reveal numerous membrane bound bacilli.



Figure 38.4 Histological appearance of a duodenal biopsy with the special stain DPAS (diastase periodic acid-Schiff). This stain highlights diastase resistant mucin within the foamy macrophages residing in the lamina propria. The mucin within goblet cells is also positively stained.